DISEASE KNOWLEDGE AND QUALITY OF LIFE FOR ADULT ASTHMATICS

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ABSTRACT

The scholars believed that the education of asthma knowledge for adult asthmatics may enhance their quality of life. However, the empirical results of the relationship between asthma knowledge and quality of life are still equivocal. The aim of this paper is to reexamine the relationship between asthma knowledge and quality of life. The survey sample included 453 adult patients of asthma. We tested the moderating effect of patient versus non-specified respondent and high- versus low- educational level. The results of the hypothetical models indicated that knowledge had a significant indirect effect on quality of life, via the mediation of self-care behavior and disease severity. Results did not support the alternative model, indicating that there was no direct effect, above and beyond the indirect effect, of knowledge on quality of life. The comparison analyses indicated that there was no moderating effect of respondent’s identity and patient’s educational level on the relationship between knowledge and quality of life. Discussions about the improvement on quality of care are also provided.

Keywords: asthma, self-care behavior, multi-group comparison, disease management
INTRODUCTION

Whether asthma knowledge can affect the quality of life (QOL) of adult asthmatics is still equivocal. Some scholars found that asthma education might improve patient QOL (Jacobs, van de Lisdonk, Smeeele, van Weel, & Grol, 2001; Leroyer, Lebrun, Proust, Lenne, Lucas, Rio, Dewitte, & Clavier, 1998; Moudgil, Marshall, & Honeybourne, 2000; Gibson, Henry, Vimpani, & Halliday, 1995) even when medication regimens are not altered (Collura-Burke, Speller-Brown, Smith, Nelson, & Schmidt, 2003), while others did not find significant improvement in QOL when a patient's asthma knowledge increased (Meszaros, Orosz, Magyar, Mesko, & Vincze, 2003; Meszaros, Vincze, Mesko, & Orosz, 2001). Since there is a growing trend in health care providers conducting asthma disease management programs (ADMP) (Baker, Middleton, & Campbell, 2003), these contradictory results will have a negative impact on resource allocation because the relative effectiveness of ADMP is not conclusive (Dickinson, Hutton, Atkin, & Jones, 1997). The aim of this paper is to examine the relationship between asthma knowledge and QOL with consideration of some factors that may affect the relationship between the two.

First, we argued that there would be a trivial direct effect, above and beyond the indirect effect, of asthma knowledge on patients' QOL. The effect of knowledge on QOL would be mainly indirect; that is, only if knowledge acts on other factors affecting QOL, then its effect may be recognized. Further, it is crucial to find pivotal variables that mediate the effect of knowledge on QOL. Psychological and physiological factors may be two key categories of variables affecting QOL. In this paper, we emphasized the physiological side and examined the mediating role of disease severity. According to general theories of behavior, such as the theory of reasoned action (Ajzen & Fishbein, 1980; Fishbein & Ajzen, 1975), theory of planned behavior (Ajzen, 1985; Ajzen & Madden, 1986), and health belief model (Rosenstock, 1966), self-care behavior (SCB) is an important factor that may embody utilities of knowledge to reduce disease severity. Results of several studies supported this perspective in part of the causal sequences (Becker, 1985; Legorreta, Leung, Berkbigler, Evans, & Liu, 2000; Weingarten, Henning, 

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**Hypothesis 1**: Asthma knowledge would have a total effect on patient's quality of life.

**Hypothesis 2**: Self-care behavior and disease severity would mediate the effect of asthma knowledge on patient's quality of life.

**Hypothesis 3a**: Asthma knowledge would have a direct effect, above and beyond the indirect effect, on patient's quality of life.

**Hypothesis 3b**: Asthma knowledge would not have a direct effect, above and beyond the indirect effect, on patient's quality of life.

The hypothesis 3a and 3b were competitive hypotheses. Though we asserted that asthma knowledge would have a trivial direct effect on quality of life, we couldn't argue it directly with a negative statement. Therefore we hypothesized for the existence of direct effect. Fail to support the hypothesis 3a will indicate support for hypothesis 3b.

Secondly, moderators may also mask the main effect observed. We suspected that the respondent's identity (patient versus non-patient respondent; please refer to the term "non-specified respondent" below and discussion) and patient's educational level may moderate the relationship between asthma knowledge and QOL.

**Hypothesis 4**: Respondent's identity (patient vs. non-patient) would moderate the relationship between asthma knowledge and patient's quality of life.

**Hypothesis 5**: Patient's educational level would moderate the relationship between asthma knowledge and patient's quality of life.

Thirdly, we argued that a course of intervention for a sufficient period might be crucial in improving QOL (Moudgil et al., 2000; Meszaros et al., 2003, 2001). Therefore, a study that was based on ADMP and prolonged for at least one year is favorable in testing such a relationship.
METHOD

Research Process and Statistical Procedures

First, we developed instruments and performed a pre-test. To avoid common method variance, we determined patients’ asthma knowledge, the key independent variable, by direct testing via questionnaire and composing the score ex post. We added an item to denote respondent’s identity (patient versus non-patient) for testing moderating effect. Second, after checking face validity, expert validity, and reliability according to the results of the pre-test, we refined the questionnaire and delivered it by mail. Third, we employed software package SPSS 10.0 and LISREL 8.80 to conduct preliminary analysis, confirmatory factor analysis (CFA) for QOL, and further statistical analysis on the returned questionnaires.

The statistical procedure is as follow. First, we examined our hypothetical model and checked its goodness of fit indices. Second, we tested the alternative model and compared it with the hypothetical model to decide which one is better. Third, we conducted multi-group comparisons to examine the moderating effect of the respondent’s identity and patient’s educational level. All these steps were conducted with LISREL 8.80.

Instruments

The questionnaire consisted of five parts: QOL, asthma knowledge, asthma-related attitude, SCB, and disease severity. Asthma-related attitude, SCB, and disease severity were self-reported via a questionnaire developed from the same documents as asthma knowledge. We also control for patient’s age and attitude of asthma. The former was a single item ordinal scale with six age groups. The latter were a self-developed scale. The details of the main variables followed.

QOL. We adopted Juniper’s (Juniper, Guyatt, Feeny, & Griffith, 1993) Chinese (Taiwan) version of the Asthma Quality of Life Questionnaire (AQLQ) to determine the asthmatics’ QOL.
Asthma knowledge. Asthma knowledge (eight items with a trichotomy scale: “yes”, “no”, or “I don’t know”) was developed using on the *Diagnostic and Therapeutic Guideline of Asthma* (2000), and *Pocket Guide for Asthma Management and Prevention in Children: A Pocket Guide for Physicians and Nurses* (2005) questionnaire. A score of eight indicated that all items were answered correctly, while zero indicated that none of the answers were correct.

Asthma-related attitude. An *attitude* high score (10 items, Likert-5 scale) indicated an initiative, positive, and highly tolerant attitude.

SCB. *SCB* (15 items, Likert-5 scale) included four dimensions: prevention of attack, proper use of medication, self-first aid for attack, and periodical monitoring of pulmonary function. The higher the score, the more serious the behavior.

Disease severity. *Severity* detected the frequency of attack during daytime and nighttime hours, and a total evaluation of severity. The respondents were asked to self-report on this scale. A high score indicated a worsening condition during the past four weeks.

Data Collection

The subjects studied were adult asthmatics who, during December 2001 and December 2002, participated in the ADMP, which has been held by the Bureau of National Health Insurance in Taiwan since November 2001. In this program, patients received intervention and education every three months. We sent 150 preliminary questionnaires by mail for pre-testing. Cronbach’s α of all constructs in the returned pre-test sample were greater than 0.70. We sent 1,595 finalized questionnaires and received 453 valid samples (28.4% response rate). Cronbach’s α of four dimensions of SCB were higher than 0.70, while attitude was 0.67.

CFA of AQLQ

The Cronbach’s α of the four dimensions of AQLQ (symptom, activities, emotion, and environment) were 0.95, 0.87, 0.87, and 0.88, respectively. Before conducting the CFA of AQLQ, we checked and deleted two items (work-related activities & sleep)
because of high percentage of missing data (21.4% & 25.2%). In determining overall
the model fit, we adopted the recommendation of Vandenberg and Lance (2000), using
RMSEA, NNFI, CFI, and SRMR as complements of chi-square test. In determining the
significance of differences between two nested models, we adopted Cheung and
Resnold's (2002) suggestion, using $\Delta$CFI < .01 as criterion. If the $\Delta$CFI is greater
then .01, the difference between two nested models is significant.

The total-item CFA model of our sample revealed an acceptable fit ($\chi^2/df =
2511.60/399$, $p < 0.000$, RMSEA = 0.128, NNFI = 0.96, CFI = 0.96, SRMR = 0.066).
Since a total-item CFA model might make a full SEM model too complex, we parcelled
the items into four factors for conducting subsequent full model. The model fit of the
four-factor CFA was also acceptable ($\chi^2/df = 73.29/2$, $p < 0.000$, RMSEA = 0.291,
NNFI = 0.89, CFI = 0.96, SRMR = 0.033).

Model Specifications

According to general theories of behavior (Ajzen, 1985; Ajzen & Madden, 1986;
Rosenstock, 1966; Ajzen & Fishbein, 1980; Fishbein & Ajzen, 1975), we proposed
SCB-severity as main causal chain between knowledge and QOL. Patient's age, which
was coded into six age groups, and attitude in our model served as control variables of
SCB and QOL. These variables and paths comprise our hypothetical model (Figure 1).

Since there is no re-test data available for estimating the reliability of knowledge,
we specified error of observed knowledge as zero, factor loading as one, and free
estimate for variance of latent knowledge. The same setting was applied to age group
based on the same reasoning. We fixed error of observed attitude as 0.12 and factor
loading as 0.82 so as to reflect the reliability (0.67) and error variance (0.39) of attitude.
We set scale for every endogenous latent construct by fixing one of the observed
variables of each construct as one. All other factor loadings, errors, and all path
coefficients were all freed for estimation.
**FIGURE 1 Hypothetical and alternative model.**
(The model with dotted line is the alternative model.)

KNOW = Asthma knowledge  
ATTI = Asthma-related attitude  
AG = Age group  
SCB1 = Prevention of attack  
SCB2 = Proper use of medication  
SCB3 = Self-first aid for attack  
SCB4 = Periodical monitoring  
SEV1 = Frequency of attack in nighttime hours  
SEV2 = Frequency of attack in daytime hours  
SEV3 = Total evaluation of severity  
QOL1 = Symptom  
QOL2 = Activities  
QOL3 = Emotion  
QOL4 = Environment  

The path settings of the *alternative* model in this paper were the same as the hypothetical model except that there is an additional path from knowledge to QOL. In another sense, the *hypothetical* model can be viewed as a nested model of the *alternative* model, with the direct path between asthma knowledge and QOL being
constrained as zero. As mentioned previously, we argued that there would be a trivial direct effect of knowledge on QOL above and beyond the indirect effect. Accordingly, a poorer fit of the alternative model than the hypothetical model or a non-significant coefficient of the direct path will indicate a fail to support the existence of the direct effect.

The path settings of the multi-group models (patient vs. non-patient, and high-education vs. low-education) were the same as the hypothetical model when examining the invariance between two groups within the hypothetical model, and the same as the alternative model when examining the invariance between two groups within the alternative model.

RESULTS

The percentage of male in our sample is 52.3%, while female is 47.7%. The p value of chi-square test between gender proportion of our sample and the frame population (47.5% and 52.0%) is 0.051, while age distribution is 0.151. Table 1 shows the demographic data of our sample. Table 2 relays the means and standard deviations of variables of the patient and non-specified respondent sample. Table 3 illustrates the correlations and reliabilities of the variables analyzed.

<table>
<thead>
<tr>
<th></th>
<th>Number</th>
<th>%</th>
<th>p-value ($\chi^2$-test)</th>
</tr>
</thead>
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<td>Gender</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>237</td>
<td>52.3</td>
<td>0.051</td>
</tr>
<tr>
<td>Female</td>
<td>216</td>
<td>47.7</td>
<td></td>
</tr>
<tr>
<td>Age group</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Unknown</td>
<td>3</td>
<td>0.7</td>
<td>0.151</td>
</tr>
<tr>
<td>18-29</td>
<td>61</td>
<td>13.5</td>
<td></td>
</tr>
<tr>
<td>30-39</td>
<td>55</td>
<td>12.1</td>
<td></td>
</tr>
<tr>
<td>40-49</td>
<td>89</td>
<td>19.7</td>
<td></td>
</tr>
<tr>
<td>50-59</td>
<td>85</td>
<td>18.8</td>
<td></td>
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<tr>
<td>&gt; 60</td>
<td>160</td>
<td>35.3</td>
<td></td>
</tr>
<tr>
<td>Respondent</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Patient</td>
<td>367</td>
<td>81.0</td>
<td>--</td>
</tr>
<tr>
<td>Non-patient</td>
<td>63</td>
<td>13.9</td>
<td></td>
</tr>
<tr>
<td>Missing</td>
<td>23</td>
<td>5.1</td>
<td></td>
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TABLE 2 Results of comparison between patient and non-specified respondent group

<table>
<thead>
<tr>
<th></th>
<th>Patient Respondent (n=367)</th>
<th>Non-specified Respondent (n=86)</th>
<th>p-value</th>
</tr>
</thead>
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<tr>
<td>Gender</td>
<td>1.45 (0.03)</td>
<td>1.54 (0.06)</td>
<td>0.169</td>
</tr>
<tr>
<td>Education</td>
<td>3.65 (0.06)</td>
<td>2.57 (0.13)</td>
<td>0.000</td>
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<tr>
<td>Knowledge</td>
<td>5.36 (0.08)</td>
<td>4.34 (0.18)</td>
<td>0.000</td>
</tr>
<tr>
<td>Attitude</td>
<td>3.06 (0.03)</td>
<td>2.91 (0.06)</td>
<td>0.020</td>
</tr>
<tr>
<td>Age Group</td>
<td>4.25 (0.08)</td>
<td>4.92 (0.15)</td>
<td>0.000</td>
</tr>
<tr>
<td>SCB1</td>
<td>4.06 (0.04)</td>
<td>3.54 (0.10)</td>
<td>0.000</td>
</tr>
<tr>
<td>SCB2</td>
<td>2.62 (0.07)</td>
<td>2.16 (0.13)</td>
<td>0.002</td>
</tr>
<tr>
<td>SCB3</td>
<td>3.74 (0.04)</td>
<td>3.24 (0.09)</td>
<td>0.000</td>
</tr>
<tr>
<td>SCB4</td>
<td>4.25 (0.04)</td>
<td>4.07 (0.11)</td>
<td>0.127</td>
</tr>
<tr>
<td>SEV1</td>
<td>2.10 (0.06)</td>
<td>2.43 (0.11)</td>
<td>0.011</td>
</tr>
<tr>
<td>SEV2</td>
<td>1.92 (0.05)</td>
<td>1.98 (0.10)</td>
<td>0.570</td>
</tr>
<tr>
<td>SEV3</td>
<td>2.20 (0.05)</td>
<td>2.34 (0.09)</td>
<td>0.156</td>
</tr>
<tr>
<td>QOL1</td>
<td>4.83 (0.07)</td>
<td>4.72 (0.14)</td>
<td>0.483</td>
</tr>
<tr>
<td>QOL2</td>
<td>4.78 (0.06)</td>
<td>4.65 (0.13)</td>
<td>0.356</td>
</tr>
<tr>
<td>QOL3</td>
<td>4.62 (0.08)</td>
<td>4.57 (0.17)</td>
<td>0.804</td>
</tr>
<tr>
<td>QOL4</td>
<td>4.58 (0.08)</td>
<td>4.68 (0.17)</td>
<td>0.579</td>
</tr>
</tbody>
</table>

*Note:* mean, (standard deviation), and p-value of t-test

SCB1 = Prevention of attack, SCB2 = Proper use of medication, SCB3 = Self-first aid for attack, SCB4 = Periodical monitoring, SEV1 = Frequency of attack in nighttime hours, SEV2 = Frequency of attack in daytime hours, SEV3 = Total evaluation of severity, QOL1 = Symptom, QOL2 = Activities, QOL3 = Emotion, QOL4 = Environment

The Hypothetical and Alternative Model

The fit indices of the *hypothetical* model showed that it was an acceptable fit ($\chi^2/df = 304.53/71$, $p = 0.000$, RMSEA = 0.087, NNFI = 0.93, CFI = 0.945, SRMR = 0.071). The total effect of asthma knowledge on QOL were 0.023 ($t = 2.71$), which indicated that knowledge makes a significant impact on QOL by means of our proposed causal chain. The hypothesis 1 was supported.
### TABLE 3 Correlation matrix of full model

<table>
<thead>
<tr>
<th></th>
<th>Knowledge</th>
<th>Attitude</th>
<th>Age Group</th>
<th>SCB1</th>
<th>SCB2</th>
<th>SCB3</th>
<th>SCB4</th>
<th>SEV1</th>
<th>SEV2</th>
<th>SEV3</th>
<th>QOL1</th>
<th>QOL2</th>
<th>QOL3</th>
<th>QOL4</th>
</tr>
</thead>
<tbody>
<tr>
<td>Knowledge</td>
<td>-</td>
<td>0.27*</td>
<td>-0.14*</td>
<td>-0.2</td>
<td>0.05</td>
<td>(0.74)</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Attitude</td>
<td>0.27*</td>
<td>0.30*</td>
<td>0.23*</td>
<td>0.30*</td>
<td>0.09</td>
<td>0.41*</td>
<td>(0.84)</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Age Group</td>
<td>-0.14*</td>
<td>0.28*</td>
<td>0.05</td>
<td>(0.42)</td>
<td>0.00</td>
<td>0.57*</td>
<td>0.21*</td>
<td>(0.82)</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>SCB1</td>
<td>0.55*</td>
<td>0.14*</td>
<td>0.21*</td>
<td>0.21*</td>
<td>0.55*</td>
<td>0.36*</td>
<td>0.38*</td>
<td>(0.89)</td>
<td>-</td>
<td>-</td>
<td>-</td>
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<tr>
<td>SCB2</td>
<td>-0.10†</td>
<td>-0.13*</td>
<td>0.08</td>
<td>0.04</td>
<td>0.04</td>
<td>-0.18</td>
<td>-0.18</td>
<td>-0.15</td>
<td>0.46*</td>
<td>0.44*</td>
<td>-</td>
<td>-</td>
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<td>-</td>
</tr>
<tr>
<td>SCB3</td>
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<td>0.01†</td>
<td>0.04</td>
<td>0.03</td>
<td>0.03</td>
<td>0.06</td>
<td>0.06</td>
<td>0.04</td>
<td>-0.42*</td>
<td>-0.41*</td>
<td>-0.48*</td>
<td>0.80*</td>
<td>(0.67)</td>
<td></td>
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<tr>
<td>SCB4</td>
<td>0.08</td>
<td>0.08</td>
<td>0.11†</td>
<td>0.12†</td>
<td>0.08</td>
<td>-0.44*</td>
<td>-0.43*</td>
<td>-0.46*</td>
<td>0.85*</td>
<td>0.74*</td>
<td>(0.87)</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>SEV1</td>
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<td>-0.10†</td>
<td>-0.07</td>
<td>-0.11†</td>
<td>-0.11†</td>
<td>0.60*</td>
<td>0.60*</td>
<td>0.60*</td>
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<tr>
<td>SEV2</td>
<td>-0.04</td>
<td>-0.19*</td>
<td>-0.07</td>
<td>-0.08</td>
<td>0.60*</td>
<td>-</td>
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<td>-</td>
<td>-</td>
</tr>
<tr>
<td>SEV3</td>
<td>-0.13*</td>
<td>-0.28*</td>
<td>-0.04</td>
<td>-0.04</td>
<td>0.04</td>
<td>-0.18</td>
<td>-0.18</td>
<td>-0.15</td>
<td>0.46*</td>
<td>0.44*</td>
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<tr>
<td>QOL1</td>
<td>0.15*</td>
<td>0.33*</td>
<td>-0.10†</td>
<td>0.11†</td>
<td>0.17*</td>
<td>0.10†</td>
<td>0.11†</td>
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<td>-0.51*</td>
<td>-0.55*</td>
<td>(0.95)</td>
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<tr>
<td>QOL2</td>
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<td>0.19*</td>
<td>0.19*</td>
<td>0.08</td>
<td>0.03</td>
<td>0.03</td>
<td>0.01</td>
<td>0.01</td>
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<td>0.01</td>
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<tr>
<td>QOL3</td>
<td>0.13*</td>
<td>0.33*</td>
<td>-0.14*</td>
<td>-0.14*</td>
<td>0.08</td>
<td>0.11†</td>
<td>0.11†</td>
<td>0.12†</td>
<td>0.08</td>
<td>0.12†</td>
<td>0.08</td>
<td>0.12†</td>
<td>0.08</td>
<td>0.12†</td>
</tr>
<tr>
<td>QOL4</td>
<td>0.13*</td>
<td>0.19*</td>
<td>-0.14*</td>
<td>-0.14*</td>
<td>0.08</td>
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<td>0.08</td>
<td>0.08</td>
<td>0.08</td>
<td>0.08</td>
<td>0.08</td>
</tr>
</tbody>
</table>

**Note:** Correlation coefficients and (Reliabilities)
- SCB1 = Prevention of attack, SCB2 = Proper use of medication, SCB3 = Self-first aid for attack, SCB4 = Periodical monitoring, SEV1 = Frequency of attack in nighttime hours, SEV2 = Frequency of attack in daytime hours, SEV3 = Total evaluation of severity, QOL1 = Symptom, QOL2 = Activities, QOL3 = Emotion, QOL4 = Environment.
- †: p < 0.10
- *: p < 0.05

### TABLE 4 Model fit of hypothetical and alternative models

<table>
<thead>
<tr>
<th>Model</th>
<th>Total</th>
<th>Indirect</th>
<th>$\chi^2$</th>
<th>df</th>
<th>p-value</th>
<th>RMSEA</th>
<th>NNFI</th>
<th>CFI</th>
<th>ΔCFI</th>
<th>SRMR</th>
<th>Comparison</th>
</tr>
</thead>
<tbody>
<tr>
<td>1  Hypothetical</td>
<td>0.023 (2.71)</td>
<td>0.023 (2.71)</td>
<td>304.53</td>
<td>71</td>
<td>&lt;0.001</td>
<td>0.087</td>
<td>0.93</td>
<td>0.945</td>
<td>0.071</td>
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</tr>
<tr>
<td>2  Alternative</td>
<td>0.030 (0.97)</td>
<td>0.023 (2.70)</td>
<td>304.47</td>
<td>70</td>
<td>&lt;0.001</td>
<td>0.088</td>
<td>0.93</td>
<td>0.945</td>
<td>0.071</td>
<td>2 vs. 1</td>
<td></td>
</tr>
</tbody>
</table>

**Note:** The number within parentheses is t-value. n = 453
### TABLE 5 Comparison between patient and non-patient respondents

<table>
<thead>
<tr>
<th>Model</th>
<th>Total</th>
<th>Indirect</th>
<th>$\chi^2$</th>
<th>df</th>
<th>p-value</th>
<th>RMSEA</th>
<th>NNFI</th>
<th>CFI</th>
<th>$\Delta$CFI</th>
<th>SRMR</th>
<th>Comparison</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 $H_{form}$</td>
<td>383.50</td>
<td>142</td>
<td>$&lt;0.001$</td>
<td>0.087</td>
<td>0.93</td>
<td>0.942</td>
<td>0.078/0.075</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2 $H_A$</td>
<td>394.37</td>
<td>150</td>
<td>$&lt;0.001$</td>
<td>0.086</td>
<td>0.93</td>
<td>0.941</td>
<td>-0.001</td>
<td>0.079/0.084</td>
<td>2 vs. 1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>7H $H_{A\Phi,(i,j)}$</td>
<td>0.018 (2.31)</td>
<td>0.018 (2.31)</td>
<td>400.34</td>
<td>156</td>
<td>$&lt;0.001$</td>
<td>0.085</td>
<td>0.93</td>
<td>0.942</td>
<td>0.001</td>
<td>0.081/0.094</td>
<td>7H vs. 2</td>
</tr>
<tr>
<td>7A $H_{A\Phi,(i,j)}$</td>
<td>0.035 (1.04)</td>
<td>0.018 (2.32)</td>
<td>400.07</td>
<td>155</td>
<td>$&lt;0.001$</td>
<td>0.085</td>
<td>0.93</td>
<td>0.941</td>
<td>-0.001</td>
<td>0.081/0.095</td>
<td>7A vs. 2</td>
</tr>
</tbody>
</table>

*Note:* The number within parentheses is $t$-value. $n = 367$ (patient) and 86 (non-patient respondent). 7H = Model 7 for hypothetical model, 7A = Model 7 for alternative model.

### TABLE 6 Comparison between high-educated and low-educated patients

<table>
<thead>
<tr>
<th>Model</th>
<th>Total</th>
<th>Indirect</th>
<th>$\chi^2$</th>
<th>df</th>
<th>p-value</th>
<th>RMSEA</th>
<th>NNFI</th>
<th>CFI</th>
<th>$\Delta$CFI</th>
<th>SRMR</th>
<th>Comparison</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 $H_{form}$</td>
<td>375.64</td>
<td>142</td>
<td>$&lt;0.001$</td>
<td>0.088</td>
<td>0.93</td>
<td>0.944</td>
<td>0.083/0.079</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2 $H_A$</td>
<td>381.57</td>
<td>150</td>
<td>$&lt;0.001$</td>
<td>0.085</td>
<td>0.93</td>
<td>0.944</td>
<td>0.000</td>
<td>0.086/0.079</td>
<td>2 vs. 1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>7H $H_{A\Phi,(i,j)}$</td>
<td>0.024 (2.68)</td>
<td>0.024 (2.68)</td>
<td>390.11</td>
<td>156</td>
<td>$&lt;0.001$</td>
<td>0.083</td>
<td>0.93</td>
<td>0.944</td>
<td>0.000</td>
<td>0.091/0.080</td>
<td>7H vs. 2</td>
</tr>
<tr>
<td>7A $H_{A\Phi,(i,j)}$</td>
<td>0.032 (0.99)</td>
<td>0.024 (3.22)</td>
<td>390.06</td>
<td>155</td>
<td>$&lt;0.001$</td>
<td>0.084</td>
<td>0.93</td>
<td>0.943</td>
<td>-0.001</td>
<td>0.092/0.080</td>
<td>7A vs. 2</td>
</tr>
</tbody>
</table>

*Note:* The number within parentheses is $t$-value. $n = 189$ (low-educated patient) and 256 (high-educated patient). 7H = Model 7 for hypothetical model, 7A = Model 7 for alternative model.
The alternative model also showed an acceptable fit ($\chi^2/df = 304.47/70$, $p = 0.000$, RMSEA = 0.088, NNFI = 0.93, CFI = 0.945, SRMR = 0.071). The indirect effect of knowledge on QOL was 0.023 ($t = 2.70$), indicating that the indirect effect hold in the alternative model. The hypothesis 2 was supported.

The $\Delta$CFI was less then .01, indicating that the two nested models were equally fit and that the constraint of the direct path between knowledge and QOL were tenable. The path coefficient of knowledge to QOL in the alternative model was non-significant (0.007, $t = 0.231$). The non-significant path coefficient further provided the evidence that the direct effect of asthma knowledge on QOL was trivial. This result indicated that knowledge did not have a significant direct effect on QOL. The hypothesis 3a was not supported and the hypothesis 3b was supported.

**Multi-group Comparison**

Cheung and Rensvold (2002) proposed a procedure for testing multi-group CFA. According to Cheung and Rensvold’s (2002) procedure, there are eight models in testing several aspects of measurement invariance. The first five models relate to measurement level invariance, whereas the last three relate to construct level invariance.

There are three steps for testing the invariance of coefficients in structural model. The first step is configural invariance (Model 1, $H_{form}$): the overall model fit of Model 1 provides evidence that all groups share the same factor structure. The second step is construct-level metric invariance (Model 2, $H_{b}$): a non-significant change in certain fit index (i.e., $\Delta$CFI < .01) from Model 1 to Model 2 provides evidence that the factor loadings between observed variables and their underlying construct are the same for both groups. The third step is equivalence of construct covariance (Model 7, $H_{A}^{(1)}$): a non-significant change in certain index (i.e., $\Delta$CFI < .01) from Model 2 to Model 7 provides evidence that the coefficients among constructs are the same across groups.

Table 5 and 6 shows the values of fit indices in each step of the group comparison for respondent’s identity and patient’s educational level, respectively. Both the two comparisons on hypothetical model passed all the three steps, indicating that all the
coefficients in the hypothetical model were invariant between the two compared group (between patient and non-patient respondents, and between high- and low- educated patients). In other words, neither the respondent’s identity nor the patient’s educational level act as a moderator on the hypothetical model.

In the comparison of the alternative model, both the two comparisons passed all three steps passed, too. The direct paths between knowledge and QOL were still non-significant in all of the comparison models. The hypothesis 4 and 5 were not supported.

**DISCUSSION**

**Impact of Asthma Knowledge on QOL**

Our study showed that the direct effect of asthma knowledge on QOL was not significant and the indirect effect was significant. The practical meaning is that knowledge does not have an additional impact on QOL above and beyond the effect of disease severity, holding attitude and age group constant. More specifically, the way asthma knowledge may act on QOL in this paper is to reduce disease severity by patient’s seriously conducting self-care behavior.

The implication of this result on the equivocal knowledge-QOL relationship among relevant studies is at least threefold. First, the content of knowledge may be a crucial factor. In our study, the effect on knowledge can never act above and beyond the effect of disease severity on QOL. This implies that, even though asthma knowledge may improve psychological well-being of asthma patient, its effects never add value to QOL. The point is that the way asthma knowledge may help the patients is to improve the physiological well-being. The knowledge for eliminating, for example, patient’s anxiety may not work for improving QOL.

Second, study design may affect the results. It is not likely that patients’ knowledge increased as their disease severity decreased simultaneously without any time lag. Thus the time lag between knowledge transfer and disease severity evaluation is therefore pivotal in detecting knowledge’s impact on QOL.
Third, the manner of asthma education may also play a role. A course of education that is designed to repeat intervention and review knowledge after a period of time may be better than an episodic style of education course. Disease management program in this regard is better than summer camping-like program.

**Moderating Role of Respondent's Identity and Patient’s Educational Level**

We were interested in whether the identity of the respondent moderated the relationships among variables or not. Our design for addressing this issue was simply to add an item to denote whether the respondent is a patient or not. This design has some advantages. First, it corresponds with a natural condition. A predetermined assignment on a respondent may upset patients if they do not have a suitable assistant to help them respond to the questionnaires, and therefore make replies more difficult to interpret. Second, it reflects a natural proportion of patients that do not respond to similar questionnaires by themselves. This proportion helps us to evaluate potential distortions if a study does not gather information on a respondent’s identity. In our study, this proportion is around 13.9-19.0 % of all respondents.

However, this design has several shortcomings. First, the authors cannot understand the reasons why patients did not answer the questionnaires themselves. Second, the author cannot control for specific identity of non-patient respondent (e.g., spokesperson, caregiver, other key person, or other family member) ex ante. Third, some questions may not clearly reflect the patient’s thought. In our study, we cannot exactly know who (patient or non-patient respondent) answer the questions of asthma knowledge if the respondent was not the patient.

In our sample, 63 respondents were not patients, while another 23 respondents did not fill out this item. We supposed that there were two kinds of respondents in these 63 respondents: (1) spokespeople for the patients who rate on all items according exactly to the patients’ thought; and (2) other people who answered some or all items based on their opinion versus that of the patients. As for the case of the former, which might simply be due to a patient being illiterate, the relationships among variables would be the same as if patient-respondents’ answered the questionnaire themselves with little
distortion by the spokesperson. As for the latter case, which may be due to various causes, the relationships among variables were invariant to the case of patient-respondents according to our study of the multi-group comparison. Though we cannot determine the ratio between these two kinds of respondents, it seems that each case would not significantly alter the relationships among the variables.

Though we do not know the respondent’s identity (patient or non-patient) in the other 23 questionnaires, their condition might be similar to the case of non-patient respondents; that is, (1) spokespeople or patients themselves, and (2) other people. Thus, we arbitrarily included these respondents into the non-patient group and called them the “non-specified respondent” group.

Since a low level of education may be the most possible cause of non-specified respondent ratings, we conducted the second two-group comparison (low versus high educational level) to investigate whether educational level moderates relationships among variables. The results show that there were invariant between the two groups in the hypothetical and alternative models, and that the direct path coefficients of knowledge to QOL were not significant.

In summary, we conclude that respondents’ identity, whether patients or not, did not significantly alter the relationship among variables in our study. Further, there was no significant evidence showed that educational level moderates relationships among variables.

CONCLUSION

Implications

There are some practical implications according to the results of this study. First, promoting asthma knowledge to patients may helpful in improving their QOL. Knowledge significantly relate to QOL in our study, though the accurate latent period of its effect is unknown. Repeated intervention is highly recommended. Furthermore, promoting asthma knowledge to patients’ families, caregivers, or other key persons may also help.
Second, the goal of promoting asthma knowledge should focus on severity reduction. Though we cannot rule out the possibility that knowledge may have an impact on variables other than the causal chain of SCB-severity, we found in the present study that knowledge is significantly related to SCB and, consequently, severity and QOL. Thus, maximizing the effect of the SCB-severity chain may markedly improve patient QOL.

Third, other efforts are also recommended to improve patient QOL. For example, changing attitude may influence QOL directly and indirectly. The significance of age group on QOL implies that older patients had lower QOL and practitioners should pay more attention to them.

As for empirical implications, we stressed on two items. First, consideration of a latent period in conducting experiment-based study is recommended to directly support the causal effect of knowledge on QOL via the impact on severity. Here we emphasize on the role of an adequate latent period, which may be a critical factor in determining the significance of relationship between knowledge and QOL. Moudgil and colleagues (Moudgil et al., 2000) illustrated the significance between intervention and QOL, which was based on a 12 month follow up study. The significant result between knowledge and QOL in our study is based on cases that participated in ADMP for one through thirteen months. Thus, the effect from increasing knowledge will never be longer than twelve months. The results of Meszaros and colleagues (Meszaros et al., 2003, 2001) are non-significant, but based on three months panel studies. Therefore, we suggest that the most beneficial time period for obtaining significant results should be between three and twelve months.

Second, it is important to investigate the real causes behind non-patient response. There are several reasons for this suggestion. First, the proportion of non-patient response was, in our opinion, too high. We cannot intuitively determine reasonable causes for this percentage. Second, it is necessary to provide direct evidence that non-patient respondents do not alter the relationship between knowledge and QOL. The invariance between models in the present study is indirect evidence. Further, a relatively small sample size in the non-specified respondent group makes it easier to
conclude that something is non-significant for a non-patient group. Direct evidence based on distinguished causes of non-patient responses may make the conclusion more robust. Third, we need to develop alternative knowledge-promoting programs that are based on various non-patient responses. Knowledge and SCB are significantly lower in the non-specified respondent group (see table 2). Exploring the causes behind non-patient ratings may help us to develop programs to increase their knowledge and SCB. Finally, it is helpful to understand why the attitude of the non-specified respondent group is lower than the others (see table 2) since attitude is a significant predictor of QOL, and why severity and QOL are not significantly lower.

Limitations

We note some limitations of this paper. First, the causal effect cannot be treated as merited because it is a cross-sectional design. The wording of causal effect is, on the one hand, statistical verbiages and, on the other hand, explanatory accommodation. We do not think that this paper established any theoretical causal relationship.

Second, the frame population of this paper, which the patients were not enrolled in randomly, restricts its external validity and, maybe, affects internal validity. The enrollment of patients into the ADMP may be influenced by the patient’s willingness to participate, physicians’ assessment of patients’ condition, and hospital administrative considerations. We have little information about the impact of these factors on our study.

Third, a fair response rate may bias internal validity. However, the chi-square test between our sample and the frame population are not significantly different in gender proportion and age distribution. Thus, we believed that the bias from the fair response rate might be trivial and limited.
REFERENCES


Biographical Sketch

Chung-Ting Lo is currently a doctoral candidate of the Graduate School of Management at I-Shou University in Taiwan. He received her B.S. degree in School of Medicine at China Medical University and M.S. degree in Graduate School of Management at I-Shou University in Taiwan. His current research interests include leadership, workplace mistreatment, and disease management.

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成人氣喘患者的知識與生活品質

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中文摘要

許多研究指出對氣喘病患施行衛教，可增進其因應疾病的能力，有助提昇其生活品質，然而實證研究所獲的證據並不一致。本研究的目的在於檢驗氣喘知識與生活品質之間的關係。我們同時也檢驗答卷者身份（病患親答或旁人代筆）及病患教育程度的調節效果。結果顯示，病患的氣喘知識透過提升自我照顧能力以降低疾病嚴重度，進而其生活品質產生顯著的間接效果；在間接效果之外，知識對生活品質並無直接效果存在。多群體比較分析顯示，答卷者身份及病患教育程度對於上述關係並無調節效果。對於生活品質的相關因素，我們亦提出相關解釋與討論。

關鍵詞：氣喘、自我照護行爲、多重群組比較、疾病管理

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